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THE EVALUATION OF AUTONOMIC RESPONSES: TOWARD A GENERAL SOLUTION

BY

JOHN I. LACEY

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By

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Antioch College, Yellow Springs, Ohio

Responses of the central and peripheral structures of the autonomic nervous system are ubiquitous. One cannot stimulate the organism, however innocuously, without producing some evidence of disturbance of autonomic equilibrium. This fact is still not thoroughly integrated with our thinking about psychological matters, but the realization is growing that restricting consideration of the autonomic nervous system to a chapter on "emotions" in our texts and reviews is an underemphasis, or at least a misemphasis.

A brief enumeration of some of the relationships found or suggested may serve to illustrate the scope and importance of the measurement of autonomic nervous system functions. The activity of the autonomic nervous system is related to the adequacy of sensory, sensorimotor, and perceptual behavior (E. Calloway and S. V. Thompson, 1953; N. Kleitman, S. Titelbaum, and P. Feiveson, 1938; A. Trehub, 1954; J. J. Van Biervliet, 1894) and to the adequacy of the performance of skilled behavior (T. A. Hussman, 1955). Measurements of autonomic responses to the innocuous stress of mild exercise (the Schneider test) are related to psychiatric status (R. A. McFarland and J. H. Huddleson, 1936), to "frustration tolerance" (G. B. Strother and D. M. Cook, 1953) and to individual differences in the relative frequency of recall of completed and incompleted tasks (T. M. Abel, 1938). Autonomic responses to injections of epinephrine and of mecholyl (the Funkenstein test) are related to recovery rate under both psychotherapy and somatotherapies (E. Gellhorn, 1953; L. Alexander, 1955). Resting autonomic measurements are related to combat performance (M. A. Wenger, 1948). Autonomic responses are being used to evaluate the effects of psychotherapy (W. N. Thetford, 1952; O. H. Mowrer, 1953) and to study the course of the therapeutic interview (A. Di Mascio, R. Boyd, M. Greenblatt, and H. C. Solomon, 1955; R. Boyd and A. Di Mascio, 1954). These responses are being used in an attempt to understand the effects of the new tranquilizers such as chlorpromazine (R. A. Schneider, 1955). As "the voice of the unconscious" and the main medi-

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ating structure in the production of psychosomatic dysfunctions, the autonomic nervous system plays a central role in psychosomatic medicine. Suggestively, the autonomic nervous system appears prominently in considerations of "energy mobilization" (E. Duffy, 1951) and the "energetics of behavior" (G. L. Freeman, 1948); and its activity has been viewed as an important part of both positive and negative feed-back circuits to the cortical cells, thus controlling, modulating, and terminating cortical activity (C. W. Darrow, 1947). In these last relations, the autonomic nervous system loses its traditional role of serving as an "objective indicator" of "subjective affect," and assumes a far more important function as a self-regulatory mechanism that is part of the organic government of behavior.

This is a broad scope indeed. In an area of such importance, careful and even anxious consideration of the problem of quantifying autonomic responses is desirable. Exploration of this problem immediately raises a basic question: In what sense are we justified in saying that individual *A*, or group *A*, is more or less reactive than individual *B*, or group *B*? Or, that individual *A* is more reactive to stimulus *a* than to stimulus *b*? The answer to this question involves at least 3 important issues: the "law of initial values" and both intraindividual and interstressor differences in the organization of autonomic responses.

The "Law of Initial Values"

The fundamental problem is to decide on the mathematics to be used. That there is a real problem here is evidenced by a large and discomforting literature on the measurement of changes in skin resistance. There are unsuspected pitfalls awaiting the investigator who simply and expediently quantifies skin-resistance responses as an algebraic or percentage change. One of the main sources of difficulty, of course, is that the magnitude of the response is related to the prestimulation level. In general, it has been found that high autonomic excitation preceding stimulation is correlated with low autonomic reactivity upon stimulation. In comparing individuals or groups, then, one does not know whether to attribute differences in obtained reactivity to pre-existing differences in the background level of autonomic excitation, or to reactivity per se. Much statistical ingenuity and labor has been expended in demonstrating this phenomenon and in suggesting and testing mathematical transformations of the raw data of skin-resistance response with the hope of arriving at a meaningful and base-free measure of skin resistance change that would be generally applicable (J. P. Seward and G. H. Seward, 1935; C. W. Darrow, 1937; O. L. Lacey and P. S. Siegel, 1949; E. A. Haggard, 1945, 1949; A. S. Paintal, 1951; D. N. Elliott and E. G. Singer, 1953; M.

A. Wenger and O. C. Irwin, 1936). A bewildering array of transformations has resulted: the logarithm of the change in conductance; the change in the logarithm of conductance; logarithm of the change in resistance; the ratio of the logarithm of the change plus a constant divided by the resting level; the percentage change in resistance; the square root of conductance; the percentage any given ohmic decrease is of the maximum ohmic decrease obtained, and others.

It is significant to note that the transformations have not been rooted in physiological fact or theory but have emerged entirely as a result of statistical manipulations. Moreover, they have turned out to be specific to the original data on which they were developed, and investigators who actually go to the trouble of testing them with newly obtained data seem likely to find that they do not work (A. S. Paintal, 1951; P. H. Venables, 1955; R. A. Champion, 1950; R. A. Terry, 1953).

Strangely enough, intensive consideration has been given to the problem only with regard to skin resistance. The same problem, however, appears in the measurement of heart rate, blood pressure, blood flow, muscle potentials, blood-sugar levels, and so on. A new and a more general solution is needed.

I begin my approach to a more general solution with the bald contention that a basic physiological error is involved in the common practice of quantifying the reactions of autonomically innervated structures by computing algebraic or percentage changes, and that additional error is committed by seeking arbitrary mathematical transformations that irrationally (by which I mean empirically, and without regard to physiological theory) remove the dependence of such computed changes upon prestimulus levels of physiological functioning in a specific experimental situation. I propose that such procedures in effect ignore the basic fact that one of the main functions of the autonomic nervous system and associated endocrine glands is to maintain a homeostatic norm. I further propose that the properties of an adequate statistical model for the evaluation of autonomic responses must correspond to the real properties of the universe of events we wish to study. In what follows, I shall first attempt to delineate these "real properties," which affect our measuring operations, and I shall then attempt to develop an adequate statistical model.

Homeostatic restraint of response. A huge number of experimental observations suggest that any induced excitation or inhibition of an autonomically innervated structure instantly initiates a series of changes that serve to nullify the disturbance. There is an important corollary which somehow has escaped being set forth in italics in discussions of psychophysiological response mechanisms. The corollary is this: *the recorded autonomic response is a function both of the induced magnitude*

of autonomic activation (as it would be seen in the absence of contrary changes) and of the promptness and vigor of secondarily induced autonomic changes that serve to restrain and limit the effects of the initial disturbance. Let us be bold about it. This corollary should be made part of the implicit background of every psychophysiological investigation; no conclusions should be drawn in such investigations without explicitly taking the corollary into account.

Darrow (C. W. Darrow, 1943), in a brilliant review that should be a classic reference for psychophysiologists, attacks the problem from a different but revealing viewpoint. "The problem," he states, "is literally to determine the weight on either side of a 'balance' when that of neither side is known." Darrow considers ways of circumventing the "mutually antagonistic action of the two branches of the autonomic nervous system" by surgical, pharmacological, and experimental-statistical techniques. He desires to find means of making valid inferences concerning the individual roles of the sympathetic and parasympathetic systems in the production of a peripheral response of a given magnitude. We desire to find means of evaluating the final resultant reaction, in the belief that it is the final resultant that is of importance to a variety of problems central in psychophysiology and psychosomatic medicine.

The 2 problems, however, are intimately related. Evidence is available that suggests that it is precisely because of certain characteristics of the "mutually antagonistic action" of the sympathetic and parasympathetic that we should expect a relationship between prestimulus physiological level and stimulus-produced physiological changes.

Homeostatic restraint of cardiovascular response. We can marshal this evidence best by an elementary review of cardiovascular dynamics, emphasizing those aspects that clarify the problem of quantifying a peripheral response. While the relevant material is available in many books and articles, a most recent authoritative presentation has been made by Heymans (C. Heymans, 1950).

As is well known, peripheral resistance and cardiac output are continuously and with great sensitivity modified to maintain arterial blood pressure within relatively narrow limits. An important part of the mechanism underlying this control lies in the aortic arch and in the carotid sinus, innervated by the so-called buffer nerves (carotid sinus nerve and cardio-aortic depressor nerve). As the arterial pressure rises, the pressure-sensitive receptors in these strategically located structures are stimulated and, through their connection with the vagus, may initiate widespread changes: a decrease in the rate of impulse formation in the sino-auricular node; a diminution of the force of contraction of the auricles and ventricles; a reduction of the duration of systole; a decrease in the

conductivity of the impulses through the different parts of the heart; and peripheral vasodilation. These changes obviously reduce cardiac output and peripheral resistance, and thus lower the blood pressure. With this compensatory lowering of blood pressure, the inverse changes occur, with the result that cardiac output and peripheral resistance increase, with a consequent rise in arterial pressure. This continuous feed-back mechanism maintains the homeostatic norm of blood pressure and heart rate.

The precision and sensitivity of the mechanism are strikingly exemplified by 3 observations: (1) in the carotid sinus of the dog, a change of pressure of as little as 1 mm. Hg is sufficient to initiate compensatory changes in blood pressure; (2) at each systole there occurs an outburst of action potentials over the carotid sinus nerves and, with each diastole, a cessation of activity, at low to moderate arterial pressures; while at high pressures the impulses form a continuous train (C. Heymans, 1950; D. W. Bronk, and G. Stella, 1932; D. W. Bronk, 1935); (3) not only is the absolute level of intrasinus pressure an adequate stimulus for the pressure receptors, but so is the rate of change of that pressure (D. W. Bronk and G. Stella, 1932).

Of utmost importance to the argument of this paper are the following observations by Samaan (A. Samaan, 1935).

(1) While the reflex regulation of cardiac output is maintained both by the vagus and the cardiac sympathetic accelerator nerves, changes in vagal tonus are far more important and significant. Thus, cardiac slowing (dogs and cats) produced by moderate vagal stimulation masked the effect of simultaneous strong cardioaccelerating stimulation. For example, to cite a typical result, if there were simultaneously applied to the peripheral cut ends of the appropriate nerves a sympathetic stimulus capable of increasing heart rate by 41 beats/minute and a vagal stimulus capable of decreasing the heart rate by 34 beats/minute, the net result is a decrease of 30 beats/minute.

(2) Sympathetic excitation *potentiates* vagal inhibition. Thus, simultaneous stimulation of the cardioaccelerator nerves or small doses of epinephrine make the heart *even more sensitive to vagal stimulation.* That epinephrine sensitizes carotid sinus mechanisms has been abundantly verified (C. W. Darrow, 1943).

(3) The vagal effect is far more prompt than the acceleratory effect. Cardiac slowing upon vagal stimulation appears at the *first beat* that follows stimulation. Cardiac acceleration, however, does not make its appearance until a minimum of 2½ seconds after the onset of stimulation, and the maximal acceleratory effect does not appear until a minimum of 7 seconds has elapsed.

Putting all these effects together—the continuous and sensitive reflex regulation; the greater importance of parasympathetic than of sympathetic control; and, most important, both the potentiation of vagal influence by simultaneous sympathetic excitation and the greater rapidity of vagal effect—we are clearly led to expect that the magnitude of the absolute or relative change that can occur in an intact organism will be negatively related to the physiological level at the onset of disturbance. Put in other terms, the more powerful influence of parasympathetic inhibitor mechanisms, and their potentiation by sympathetic excitation, suggest the following interpretation of the dynamics of a cardiovascular response. As the prestimulus level of functioning increases, there is a disproportionately greater homeostatic restraint, both in increased magnitude and decreased latency and, as the magnitude of induced activation increases, there is a disproportionately greater increment of counter-reaction.

Homeostatic restraint in other organ systems. Such effects probably are not limited to the cardiovascular system. The secretion of epinephrine is self-limiting; the presence of increasing amounts of circulating epinephrine in the blood increasingly inhibits the secretion of further epinephrine and inhibits transmission across ganglionic synapses (A. Marrazzi, 1939). In a similar manner, circulating epinephrine limits the reactivity of other sympathetic functions (C. W. Darrow, 1943), including sweat-gland reactivity. The carotid sinus mechanism itself extends control over a wide variety of somatic functions. Literature reviewed by Darrow (C. W. Darrow, 1943) shows that increased intracarotid pressure blocks respiration, halts shivering, reduces or eliminates skeletal reflexes, and decreases muscle action potentials.

Many details need to be worked out, indeed, but enough is at hand to suggest that the negative relationship of stimulus-induced physiological change to prestimulus physiological level is not simply an arithmetical artifact of a restricted range of response left available to the organism whose prestimulus functioning is high. It is a *phenomenon of physiological homeostasis* and must be dealt with as such. If statistical transformations are sought in order to derive base-free measures of response, they must not be arbitrary ones. They must be related rationally to the physiological phenomenon.

Experimental observations in intact organisms. The theory outlined above is not completely unchallenged by experimental data. That homeostatic restraint of response may fail to be effective on occasion is only to be expected. Very recently, Brown and his collaborators have reported experiments on anesthetized dogs that show little or no effect of carotid sinus reflexes in moderating systolic blood-pressure response to injected

epinephrine, norepinephrine, and histamine (G. C. Boxill and R. V. Brown, 1953; R. V. Brown and J. G. Hilton, 1954; J. G. Hilton and R. V. Brown, 1954, 1955). It becomes important, then, for the psychophysiologist to test the generality of occurrence of the phenomena of homeostatic restraint in intact human organisms.

The first approach to a general recognition of the relation of autonomic changes to prestimulation level was made by Wilder (J. Wilder, 1950) in a paper that has appeared only, I believe, as an abstract. Wilder gave the phenomenon a useful name: the "law of initial values." Working, apparently, with a variety of pharmacologic agents in serial experiments with man, Wilder found in "75 to 85 per cent of the experiments" that the magnitude of increase of physiological changes decreases as the initial level increases. Indeed, at high initial levels he often found no increase and "quite often" a "paradoxical" decrease. These are significant observations, and we have verified them repeatedly in our own laboratory. A technique of quantifying autonomic responses certainly should not obscure these phenomena.

Wilder also noted another significant fact that, with inhibitory agents, the opposite rule held. The higher the initial level of autonomic excitation, the greater the decrease in the physiological function under examination.

How well does this "law" of initial values hold up? In TABLE 1 are presented the results of several of our studies involving a variety of populations, stresses, and physiological variables. The details of the experiments are published elsewhere (J. I. Lacey and R. Van Lehn, 1952; J. I. Lacey, D. E. Bateman, and R. Van Lehn, 1953). In TABLE 1 the children, subjects in the Fels longitudinal study of growth and development, range in age from 6 to 18, and are of both sexes. The mothers, also subjects in the longitudinal study, range in age from 23 to 57. The college males are a much more homogeneous group, being freshmen at Antioch College, Yellow Springs, Ohio. Our technique involves first a 15-minute period of relaxation to stabilize physiological levels. The subject is then warned that in 1 minute he will have to put his foot into a cold-water bath, or will have to do mental arithmetic, or hyperventilation, or letter association. This results in an increment of autonomic activity which we call the alerting reaction. After 1 minute of such anticipation, the subject is then required to undergo the indicated stress. Another increment of activity results that we call the stress reaction. The subject is then allowed to recover to his previous resting level before the next alerting period begins, and so on *seriatim*. For college males, there was no alerting period.

One characteristic of our treatment of data should be noted. Each physiological variable differs, of course, in its mode of response. These

Table 1

Linear Correlations Between Initial Level and Both Percentage (R_1) and Algebraic (R_2) Increments (Decimal Points Are Omitted for All Correlations)

Group	Stress	Reaction	Physiological Variable: Systolic blood pressure			Diastolic blood pressure			Palmar conductance			Heart rate			Variability of heart rate		
			N	r_1	r_2	N	r_1	r_2	N	r_1	r_2	N	r_1	r_2	N	r_1	r_2
Children	Cold pressor	Alerting	114	−09	−03	102	−27†	−21*	114	05	57‡	107	−42‡	−32‡	108	−54‡	−45‡
		Stress	98	−31†	−23*	87	−51‡	−40‡	100	−40‡	02	93	−10	−06	93	−44‡	−43‡
Mothers	Cold pressor	Alerting	40	−19	−06	40	−46‡	−30*	40	09	29	34	−39**	−25	33	−60‡	−40**
		Stress	40	05	18	40	−10	−01	80	−15	01	68	−46‡	−36†	64	−36†	−33†
Mothers	Mental arith.	Alerting	40	00	10	40	−15	−01	42	−06	07	42	−07	13	42	−30*	−28
		Stress	40	−02	18	40	−39†	−21	42	−25	−11	42	−37**	−21	42	−41†	−50†
Mothers	Letter assoc.	Alerting	40	12	23	40	−12	02	80	−16	−01	71	−32†	−13	71	−38†	−20
		Stress	40	04	16	40	−27	−17	79	−22*	03	68	−30**	−15	65	−28*	−28*
College males	Mental arith.	Total	–	–	–	–	–	–	85	−35†	31†	85	−60‡	−38‡	85	−61‡	−38‡
College males	Hyperventilation	Total	–	–	–	–	–	–	85	−12	50‡	85	−45‡	−22*	85	−51‡	−58‡
College males	Letter assoc.	Total	–	–	–	–	–	–	85	−15	46‡	85	−41‡	−19	85	−53‡	−28†
College males	Cold pressor	Total	–	–	–	–	–	–	85	−09	55‡	85	−54‡	−30†	85	−49‡	−31†

*Significant at 0.05 level.
**Significant at 0.02 level.
†Significant at 0.01 level.
‡Significant at 0.001 level.

variables have different latencies. One may appear as a smooth monophasic response, another as a polyphasic response, *etc.* To put all variables on an equal footing, and to get clearly interpretable data, we measure the *maximum sympatheticlike activity*. Thus, for heart-rate alerting reaction, we measure and average the 6 fastest heart cycles in the last minute of rest, and then the 6 fastest heart cycles in the minute of anticipation of stress. For heart-rate stress reaction, the latter figure is compared with the 6 fastest heart rates during stress.

I shall shortly attack the use of correlation coefficients in this situation, and shall show that they give an inadequate, and occasionally an erroneous, picture of the true state of affairs. With this proviso, one can expediently get some impression of the generality of the law of initial values. Considerable sampling variation is evident in TABLE 1, especially and surprisingly for systolic blood pressure (but compare the findings of Brown *et al.*, cited above). There is no room for doubt, however, that initial level is negatively related to percentage change over a wide variety of stresses and populations. The same relationship holds for algebraic change, with one startling exception. For palmar conductance (measured in log micro-ohms), we secure significant positive correlations. One possible reason for this will become apparent in the discussion below concerning the complexities of meaning of correlation coefficients between initial level and a measure of change. Before leaving this table, it is well to note that these are linear correlations, and that bivariate normality is not assured. In many instances (see below), curvilinear correlations would reveal relations of increased magnitude.

In FIGURES 1 and 2 are shown the results of serial experiments with college males. These are subjects participating in an hour-long session devoted to the study of semantic conditioning and generalization of unconscious anxiety (J. I. Lacey and R. L. Smith, 1954; J. I. Lacey, R. L. Smith, and A. Green, 1955). Heart-rate responses repeatedly are elicited at 45 second intervals by combined motor activity (tapping a telegraph key) and verbal activity (chain association to stimulus words). For 28 of such episodes of reaction spread through the hour, the coefficient of correlation between initial value (6 fastest cardiac cycles during the last 15 seconds of a 30-second intertrial rest period) and change to stress level (6 fastest cardiac cycles during 15 seconds of verbal-motor activity) was computed for each of 21 subjects. The results again strongly support the law of initial values.

The Use of Regression Models

What can we do about this dependence of change upon initial level? Desirable and rational experimental arrangements are obvious. In

serial experiments with man, impose each succeeding stressful episode upon the same initial level. This is possible, but many times extremely difficult to attain. In group experiments, allow each person to relax or recover to a predetermined level, constant for all subjects, before administering the stress. This is manifestly impossible and, in itself, it is

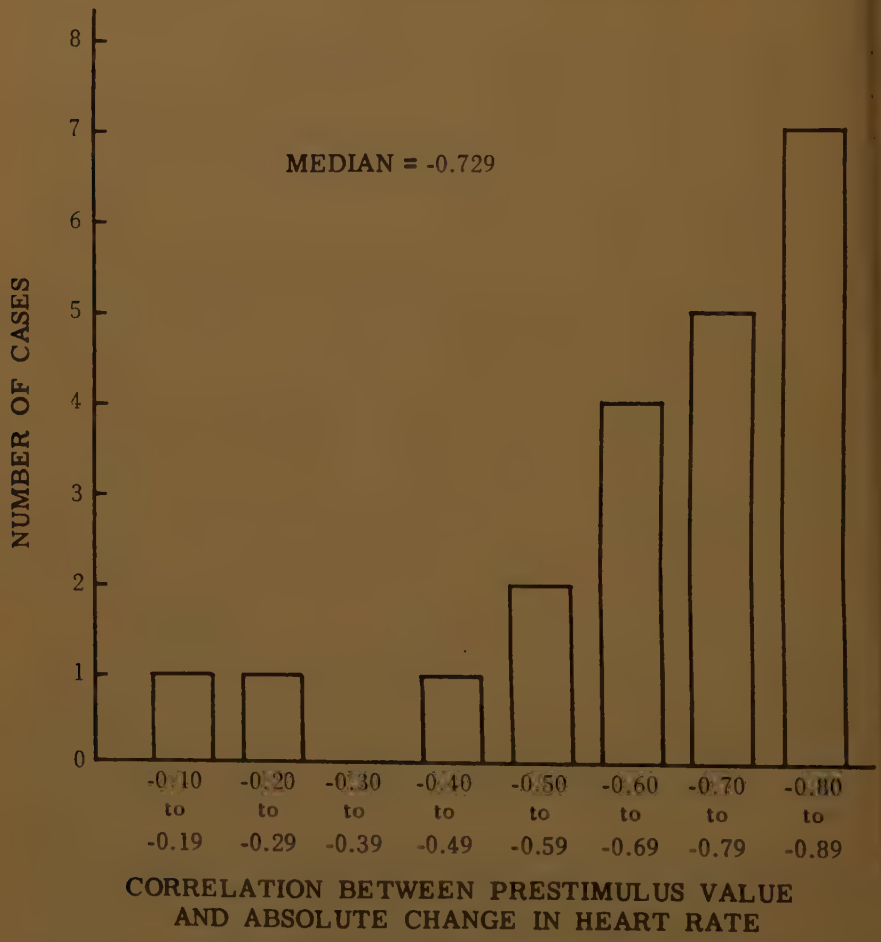

FIGURE 1. Frequency distribution for 21 cases of the individual product-moment coefficients of correlation between prestimulus heart rates and the algebraic increments of heart rate due to combined verbal-motor activity. The cardiac activity during each episode of stimulation is measured by averaging the 6 fastest cardiac rates in 15 seconds of combined activity. From this value is subtracted the average of the 6 fastest cardiac rates in the last 15 seconds of the preceding 30-second intertrial rest period, to yield the algebraic increment in "maximum sympatheticlike activity." Each correlation is based on 28 serially elicited reactions. Note that all correlations are negative, and that the median correlation is −0.73.

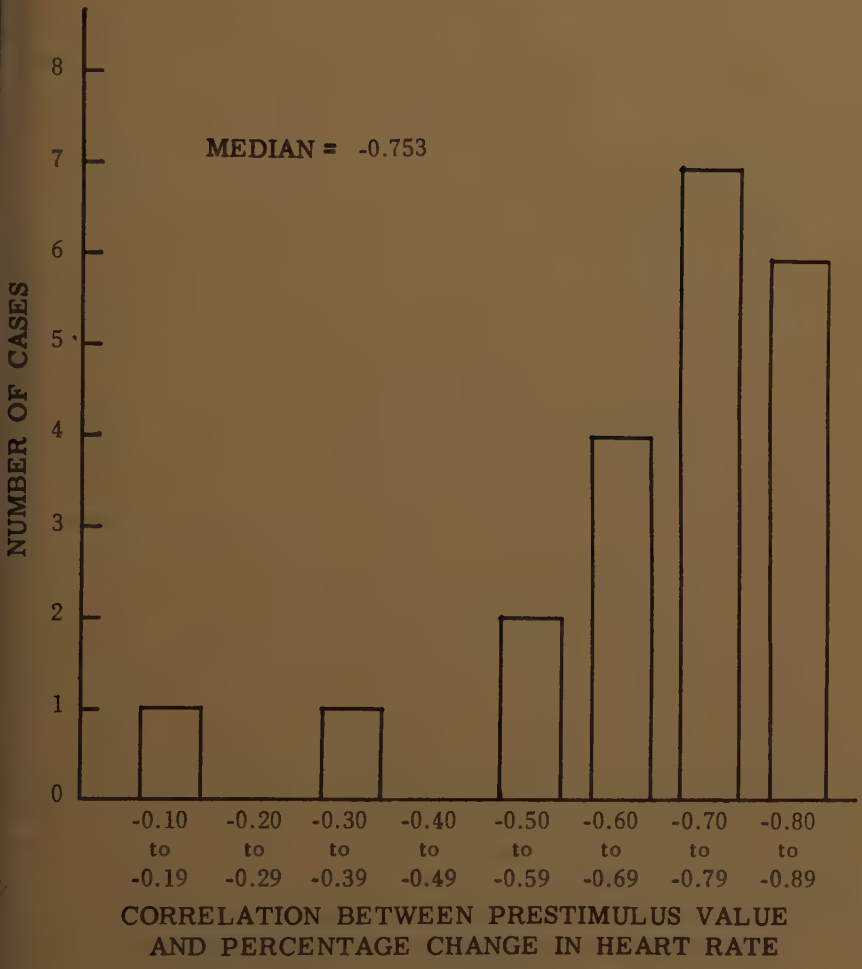

FIGURE 2. Frequency distribution for 21 cases of the individual product-moment coefficients of correlation between prestimulus heart rates and the percentage increments of heart rate due to combined verbal-motor activity. Prestimulus and stimulated levels of heart rate are determined as in FIGURE 1. Each correlation is again based on 28 serially elicited reactions. Note that all correlations are negative, and that the median correlation is −0.75.

technique that poses interpretative difficulties. The ideal solution would be to have extensive statistical norms for varying and well-defined populations and stresses. These norms would present separate frequency distributions of changes for varying initial levels. A given individual's response, then, could be referred to the frequency distribution of re-

sponses obtained at an initial value identical with that shown by the individual being studied, and his response could be expressed as the number of standard deviations away from the mean response. This ideal, of course, will never be attained with any broad generality. Given a well-defined experimental situation, however, and a not too prohibitively large sample of a well-defined population, we can attain a satisfactory and useful approximation to this ideal solution by utilizing statistical techniques of so-called regression analysis. A best-fitting curve, in the least-squares sense, relating the x-arrays (initial levels) to the means of the y-arrays (responses), becomes the locus of estimated average percentage or average algebraic changes at varying initial levels, and the standard deviation about this "line of regression" is an estimate of the standard deviation for any distribution of responses at any initial level.

If the data behave in certain lawful and simple ways, and if certain restrictive assumptions are met, sensitive and fairly precise inferences can be drawn, using only a knowledge of the means and standard deviations of the initial levels and the responses of our sample, and the correlation between initial level and response. What are the requirements (beyond adequate sampling of a defined population)?

First, the best-fitting curve should be linear. If it is not, and a curvilinear relationship fits the data significantly better than a linear one, we should be faced with the arduous and difficult task of deciding exactly what the form of the equation is and the equally arduous task of deciding on the parameters and constants of the equation. If we attempt to avoid this by first grouping our subjects into arrays of varying initial levels and then computing means and standard deviations for each such subgroup of individuals, we shall be losing considerable sensitivity and ease of application for the following reasons.

For the linear case, we have the simple and well-known result that $y'_z = r_{xy}x_z$, where y'_z is the y-value on the regression line that corresponds to a given x_z value (as the z subscript implies, x and y values are measured in units of sigma deviations for the entire sample), and r_{xy} is the product-moment correlation between initial level and response. That is, knowing an individual's initial level, we can solve the equation to estimate the theoretical average response at that initial level. This regression equation can be derived from ungrouped data, and we can make our inferences sensitive within one-integer class-intervals of initial level. That is, we can estimate mean response for individuals with initial levels of heart rate of 80 beats/minute and a different mean response for individuals with initial levels of 81 beats/minute, and so on. The only limitation lies in the sensitivity and precision of the original data. In the curvilinear case, however, no such simple result is likely to occur.

We shall not, in general, have a simple or rationally acceptable regression equation to solve; because of this we shall have to resort to coarse grouping of initial levels, and we shall have to compute means and standard deviations of the responses for each gross class-interval of initial level. The lack of precision and reliability of such a procedure is obvious.

Second, the curve should be homoscedastic. That is, the standard deviation of responses should be the same from x-array to x-array, varying only at random and within the limits of sampling error. If the curve is both linear and homoscedastic, we again have a simple and well-known result: the standard deviation of responses, at any initial level, is given by $\sigma_y \sqrt{1 - r_{xy}^2}$, where σ_y is the standard deviation of the total distribution of y (the refinement of securing a completely unbiased estimate by multiplying $\sigma_y \sqrt{1-r^2}$ by $\sqrt{(N-1)/(N-2)}$ is unnecessary for reasonably large N's).

Third, the y variable should be normally distributed over all x-arrays and within each x-array.

These conditions of linearity of regression, homoscedasticity, and normality, then, define the situation where the product-moment coefficient of correlation is maximally useful.

In the course of these investigations I tested the assumptions of linearity, homoscedasticity, and normality in 306 separate plots involving all the data in TABLE 1 and additional data. I consider the results of only historical interest because, as I shall show shortly, regression models involving percentage or absolute change are inappropriate, and they contain hidden traps. Nevertheless, they are worth summarizing.

(1) The normality assumption is frequently violated. Not only are distributions of absolute change or relative change not normal, but frequently no empirical transformation can be found that will normalize the distributions. Sixty per cent of the distributions of percentage change were nonnormally distributed. Approximately 40 per cent of the distributions of algebraic change were nonnormal.

(2) Moreover, the regression of response upon the initial level is often decisively nonlinear. Approximately 16 per cent of the plots show significant curvilinearity.

(3) The assumption of equal variance in the x-arrays was acceptable in every plot where normality and linearity were also satisfied.

On these grounds alone we should reject the applicability of regression models involving measurements of change, but there are even more convincing reasons for abandoning the approach. Interestingly enough, a consideration of these reasons directly suggests the appropriate and workable approach.

Regression techniques involving percentage or algebraic change involve (1) redundancy, and (2) "spurious" correlations.

Redundancy. The element of redundancy is obvious. Consider a subgroup of individuals at a given initial level, for example, at 110 mm. Hg blood pressure. Subject them to a stressor agent and record the maximum blood pressure reading. *Since all individuals have started with the same initial level, we get no more information by computing percentage or algebraic increment from this initial level as a base than we get by simply looking at the maximum reading itself.* This consideration suggests that the most direct and meaningful technique is to remove the regression of stress *level* upon base *level.* No intermediate calculation of change is required.

"Spuriousness." Let x be the initial level and y be the stress level. Then $\lambda = (y\text{-}x)$ is the algebraic increment of autonomic activity, and the ratio $\theta = (y\text{-}x)/x$ is the proportional increment. In correlating x with $(y\text{-}x)$ or x with $(y\text{-}x)/x$, the element x is common to both variables being correlated. Some intriguing and dangerous complications follow this introduction of "spurious" communality. As shown in the appendix to this paper, written by Edmund Churchill, the correlations between initial level and either percentage or algebraic change are directly a function of the correlation between initial level and stress level, and of the variances of those levels.

For algebraic change, an exact identity is given by

$$r^2_{x\lambda} = 1 - \frac{\sigma_y{}^2 (1 - r^2_{xy})}{\sigma_y{}^2 - 2 r_{xy} \sigma_y \sigma_x + \sigma_x{}^2},$$ where the symbols have their usual meaning.

Note some consequences. If $\sigma_x{}^2 = \sigma_y{}^2$, and the basic correlation between x and y is 0, then $r_{x\lambda}$ will be equal to $-.707$! In other words, even if there is no correlation of initial level with stress level, we shall get some correlation of initial level with algebraic change, depending upon the relationship of the standard deviations of the 2 levels. As the ratio of the component standard deviations varies, so will the correlations. If the ratio σ_x/σ_y is greater than r_{xy}, the correlation of x with $(y\text{-}x)$ will be negative; given the same value for r_{xy}, the correlation of x with $(y\text{-}x)$ will be positive if σ_x/σ_y is less than r_{xy}. The resultant base-change correlations are then extremely difficult to interpret, being a result simultaneously of the correlation between the component measures of level, and of the relationship between their standard deviations. The situation is far too complex to allow a simple and meaningful physiological interpretation.

For percentage change, an approximate identity is given by

$$r_{x\theta} = \frac{\pm 1\ \sqrt{1+3V^2}}{\sqrt{\frac{1+8V^2}{1+3V^2}+\frac{1-r_{xy}^2}{\left[\frac{V}{W}-r_{xy}\right]^2}}}$$

, where x, y, and θ are defined as above, V is the coefficient of variation of x, and W is the coefficient of variation of y. Here the complexities are of the same kind, except that a dominant role is played by the coefficient of variation, rather than the standard deviation, $r_{x\theta}$ taking on various values as a function of the relationship between the ratio of the 2 coefficients of variation, and the basic correlation r_{xy}, that is, as a function of $[(V/W) - r]$. Both the sign and the value of the correlation between x and θ depend on the relative value of r_{xy} and the ratio of the 2 coefficients of variation. Some of the misleading consequences of this situation are detailed in the appendix.

Let us summarize. The basic data are the initial level and the stress level. Computing percentage or absolute change is then a redundant step. Computing the correlation between such changes and the initial level introduces a maze of undesirable complexities.

We are then left with the simple recourse of dealing directly with the invariably high positive relationship (found for the group, not, as we shall see below, for the individual) between initial level and stress level. For the same data where so much difficulty was found in establishing satisfactory conditions for the use of the regression model using percentage or absolute change, I found no obstacle in this simpler and more direct application. All distributions of both initial and stress level were normal, by statistical test, either in their raw form, or by using the biologically familiar and defensible logarithmic transformation (J. H. Gaddum, 1945). All bivariate distributions yielded linear regressions and homoscedasticity by appropriate statistical tests.

Our proposed general measure of autonomic reaction, then, is simply attained, by repetitive solution, for each individual, of the equation:

$$\text{Autonomic Lability Score} = 50 + 10\left[\frac{y_z - x_z\, r_{xy}}{(1-r_{xy}^2)^{1/2}}\right]$$

, where x_z and y_z are the individual's initial and stress levels, respectively, expressed in units of sigma deviation in the total sample, r_{xy} is the correlation for the sample between initial level and stress level, and the constants 10 and 50 translate the resulting scores to a distribution with a mean of 50 and a standard deviation of 10. This last step is simply an arithmetic convenience in that it dispenses with negative numbers and provides a

system of units that makes it easy to compare an individual's reactions in any number of physiological variables simultaneously.

The interpretation of the final result is clear. Consider an individual whose autonomic lability score for blood-pressure response to the cold-pressor test is 50. The meaning is that the level which blood pressure reached during stress was exactly that level predicted from his initial level. If his response were 60, the meaning would be that the level his blood pressure reached during stress was 1 standard deviation (theoretically estimated) above the mean level (theoretically estimated) attained by those of his peers who had the same initial level.

Autonomic Lability Scores: Some Results and Problems

Certain important characteristics of the scores should be summarized:

First, they are, of course, free of dependence on base level, and are normally distributed if the assumptions underlying regression analysis are fulfilled (as they have been to date in our studies).

Second, in accordance with physiological theory, the regression line may predict a decrease in level or no change in level when additional autonomic activation is induced upon a prestimulus background of high excitation.

Third, equal absolute or percentage changes (if we compute them), starting from different initial levels, do not receive equal autonomic lability scores. In accordance with the demands of physiological theory, a given change gets larger and larger autonomic lability scores as the initial level increases, the amount by which the autonomic lability scores increase being determined by the correlation that reveals the degree of homeostatic restraint for the given variable, population, and stress. These points are illustrated in TABLES 2 and 3, using data for heart-rate responses obtained serially in man.

Fourth, at zero correlation between initial level and stress level (a result we have sometimes obtained in serial experiments), the autonomic lability score is identical, except for the translation to a distribution with a mean of 50 and a sigma of 10, with the sigma score for stress level. This, we feel, is as it should be. If there is no homeostatic relation to take into account, then the level of activation under stress is the only important variable. As the correlation between initial level and stress level increases, the autonomic lability score is increasingly divergent from the corresponding sigma score for stress level.

In general, autonomic lability scores correlate well with absolute or percentage change per se. In one study (J. I. Lacey, D. E. Bateman, and R. Van Lehn, 1953), we obtained correlations ranging from 0.74 to 0.93

TABLE 2

DATA ILLUSTRATING THE COMPUTATION AND USE OF AUTONOMIC LABILITY SCORES, USING THE HEART-RATE RESPONSES OF 57 COLLEGE MALES RESPONDING TO THE ORAL PRESENTATION OF A STIMULUS-WORD WITH A 15-SECOND PERIOD OF CHAIN ASSOCIATION AND TAPPING OF A TELEGRAPH KEY

Subject number	*Heart rate*		*Absolute change*	*Percentage change*	$50+10\left[\frac{y_z - x_z r_{xy}}{\sqrt{1-r^2}}\right]$	*Autonomic lability score*
	Prestimulus	*Stress*				
26	55.9	64.5	+8.6 +0.06215	+15.4 +3.6	$50 + 10\left[\frac{-1.499 - (-1.622)(+0.9386)}{0.3450}\right]$ =	51
22	131.3	139.9	+8.6 +0.02756	+6.5 +1.3	$50 + 10\left[\frac{+2.465 - (+2.199)(+0.9386)}{0.3450}\right]$ =	62
25	114.6	116.0	+1.4 +0.00528	+1.2 +0.3	$50 + 10\left[\frac{+1.506 - (+1.591)(+0.9386)}{0.3450}\right]$ =	50
21	124.3	125.7	+1.4 +0.00487	+1.1 +0.2	$50 + 10\left[\frac{+1.917 - (+1.954)(+0.9386)}{0.3450}\right]$ =	52
12	74.6	80.9	+6.3 +0.03521	+8.4 +1.9	$50 + 10\left[\frac{-0.339 - (-0.331)(+0.9386)}{0.3450}\right]$ =	49
27	91.4	99.1	+7.7 +0.03512	+8.4 +1.8	$50 + 10\left[\frac{+0.700 - (+0.578)(+0.9386)}{0.3450}\right]$ =	55

See text for a fuller description of the experiment. The distribution of heart rates was log normal. The average prestimulus heart rate was 1.90481, with a standard deviation of 0.09705. The average stress level of heart rate was 1.93674, with a standard deviation of 0.08482. The correlation between prestimulus and stress levels was +0.9386. Figures are carried out to several decimal places, not in a pretense of high precision of measurement, but to avoid rounding until the final calculation. This precaution seems necessary to secure good distributions of autonomic lability scores. Changes are expressed, in columns 4 and 5, using both heart rates (top entries) and log heart rates (bottom entries). Autonomic lability scores were computed using the log transformation of heart-rate distributions. Note that equivalent absolute and percentage changes receive differing autonomic lability scores, depending upon prestimulus levels, in accordance with the theory of homeostatic restraint of response.

TABLE 3

SELECTED DATA FROM A SINGLE SUBJECT, RESPONDING SERIALLY A TOTAL OF 52 TIMES, WITH VARYING PRESTIMULUS LEVELS OF HEART RATE

Heart rate		*Absolute change*	*Percentage change*	$50+10\left[\frac{y_z - x_z r_{xy}}{\sqrt{1-r^2}}\right]$		*Autonomic lability score*
Prestimulus	*Stress*					
98.0	97.6	−0.4	−0.4	$50+10\left[\frac{+0.74-(+0.98)(+0.7100)}{0.7042}\right]$	=	51
107.0	105.8	−1.2	−1.1	$50+10\left[\frac{+1.66-(+1.50)(+0.7100)}{0.7042}\right]$	=	58
107.5	105.0	−2.5	−2.3	$50+10\left[\frac{+1.50-(+1.66)(+0.7100)}{0.7042}\right]$	=	55
114.9	112.7	−2.2	−1.9	$50+10\left[\frac{+2.34-(+2.34)(+0.7100)}{0.7042}\right]$	=	60
80.6	88.0	+7.4	+9.2	$50+10\left[\frac{-0.67-(-0.98)(+0.7100)}{0.7042}\right]$	=	50
86.0	93.4	+7.4	+8.6	$50+10\left[\frac{+0.07-(-0.37)(+0.7100)}{0.7042}\right]$	=	55
96.8	104.2	+7.4	+7.6	$50+10\left[\frac{+1.36-(+0.84)(+0.7100)}{0.7042}\right]$	=	61
80.6	88.0	+7.4	+9.2	$50+10\left[\frac{-0.67-(-0.98)(+0.7100)}{0.7042}\right]$	=	50
83.0	90.6	+7.6	+9.2	$50+10\left[\frac{-0.32-(-0.74)(+0.7100)}{0.7042}\right]$	=	53

The correlation between prestimulus and stimulus levels of heart rate was +0.71. Means and standard deviations for prestimulus and stress levels of heart rate are not given, since, as is explained later in the text, scores in sigma units were directly assigned using McCall's normalizing transformation. Note the 4 occasions where the stress of verbal-motor activity produced a decrease in heart rate, when prestimulus rate was high. On each of these occasions, however, the regression line for this individual predicted even larger decreases than those obtained. The autonomic lability scores, therefore, are all above 50. Statistical predictions of no change or a decrement occur commonly at high prestimulus levels if the standard deviation under stress is less than the prestimulus standard deviation.

with a median value in the high 80's. These are high correlations, and one might wonder if the game were worth the candle. One example will serve to show how misleading absolute or percentage changes can be, and how illuminating autonomic lability scores can be.

In one unpublished study, we investigated maturational changes in patterns of autonomic activation by the cold-pressor test, using both algebraic and percentage change, for a sample of 64 children ranging in age from 6 to 18 years. We established some tidy results. For example, younger children tended to overreact in diastolic blood pressure and to underreact in heart rate, whereas older children showed the reverse pattern.

The correlation with age of a pattern-index score (defined in the reference given immediately above), indicating the degree to which diastolic blood-pressure reactivity exceeded heart-rate reactivity, was −0.60, using a percentage measure of change and −0.43, using an algebraic measure of change. Confidence levels were obviously very satisfactory. Our confidence, however, was shaken when we found in our own data the marked age changes in resting level that other investigators have found. As children grow older, their diastolic blood pressure increases and their heart rate decreases. As children grow older, then, we might expect (on the intriguing hypothesis that longitudinal experiments over a period of years will reveal, in the individual, the same result seen in short-time serial experiments) that it would be harder to evoke a marked increase in blood pressure, but easier to evoke a marked increase in heart rate. When we used autonomic lability scores, the alleged age change in the pattern of autonomic reactivity disappeared completely, and the correlation with age dropped to 0.07. The age change in prestimulus level, then, completely accounted for the apparent change in reactivity.

It is apparent, therefore, that although the correlation may be high between absolute or percentage change on the one hand and autonomic lability scores on the other, enough variance in the data remains to be attributed to base level to mislead us in interpretation. Our results to date indicate that whenever autonomic lability scores show a significant variation with our independent variable (age, sex, intensity of shock, reinforcement schedule, Rorschach responses, and the like), absolute and percentage changes do also. The autonomic lability scores, however, have always yielded clearer trends and greater interpretive clarity. On the other hand, absolute or percentage changes may yield results that do not hold up when the effect of base level is removed, as in the example just given.

Intraindividual autonomic lability scores. We attach special interest

and importance to the technique of regression analysis applied to serial experiments with the single individual, and we have successfully applied intraindividual autonomic lability scores in studies of semantic conditioning (J. I. Lacey and R. L. Smith, 1954; J. I. Lacey, R. L. Smith, and A. Green, 1955).

Certain special problems, however, arise in this application. Our experiments, in the citations already given, involved only 28 serially elicited responses. This yields an inadequate number of degrees of freedom to enable proper evaluation of normality, linearity, and homoscedasticity. Inspection of each case, however, seemed to reveal acceptable properties, and we simply went ahead with the analysis. In current experiments on the same problem we have 52 serially elicited responses, and we shall soon be in a position to make appropriate statistical tests. In the meantime other problems have arisen, and one intriguing and significant by-product has appeared.

The problem is: frequency distributions of initial levels and of stress levels for individuals sometimes are markedly nonnormal. For these individuals, the usually trustworthy logarithmic transformation failed, but a square-root transformation worked. For others, no reasonable or simple transformation would work. Such individuals remain a puzzle to us. To bypass the difficulty, we have resorted to "artificial" normalization by means of McCall's T-scores (P. O. Johnson, 1949). While not overly pleased with this somewhat irrational maneuver, the results were clear and reasonable in that the final experimental results for individuals so treated do not differ from those secured from individuals whose distributions are transformable to the normal form.

This procedure, however, recommended itself as an expedient one to use for all individuals, since it would remove the necessity of laboriously testing for normality of distribution in each individual case. No information seemed to exist, however, on the accuracy of such "artificial" normalization, especially as a function of the size of the sample. In a previous study (J. I. Lacey, D. E. Bateman and R. Van Lehn, 1953), we showed that the autonomic lability scores using the McCall transformation correlated from 0.91 to 0.99 with the scores resulting from the use of logarithmically normalized data, for an N of 85. Since then we have undertaken an empirical study, using hypothetical and actual data for heart rate, blood flow, and palmar conductance variables for which it was known that logarithmic or square-root transformations were effective. The nature of the distributions may be seen in TABLES 4 and 5. The degrees of skewness vary over a wide range, and of kurtosis over a narrow range, characteristic of the type of data with which we deal. As can be seen from the tables, the raw distributions were such that the ratio g_1/SE_{g_1}

TABLE 4

DEGREES OF SKEWNESS (G_1) AND KURTOSIS (G_2) FOR HYPOTHETICAL AND ACTUAL DISTRIBUTIONS USED TO COMPARE MCCALL'S T-SCORING METHOD WITH A NORMALIZING SQUARE-ROOT TRANSFORMATION*

	Untransformed distribution				*Transformed distribution*			
N	g_1	g_2	g_1/SE_{g_1}	g_2/SE_{g_2}	g_1	g_2	g_1/SE_{g_1}	g_2/SE_{g_2}
10	1.6	2.4	2.4	1.8	1.2	1.2	1.8	0.9
20	1.4	1.8	2.7	1.8	0.9	0.8	1.8	0.8
30	0.9	0.1	2.1	0.1	0.5	−0.6	1.3	−0.8
40	1.1	0.4	2.8	0.6	0.7	0.3	1.8	0.3
60	0.6	−0.1	2.1	−0.2	0.1	−0.6	0.4	−1.1
120	0.5	−0.1	2.4	−0.2	0.1	−0.7	0.3	−1.6

*For convenience of presentation, the figures are rounded to 1 decimal place.

enables rejection of the hypotheses that the distributions are normal, whereas the transformed distributions are acceptably normal (P. O. Johnson, 1949). From these distributions 2 sets of scores were constructed. One set of scores, in T-score form (with a mean of 50 and a standard deviation of 10), was constructed using the mathematical transformation that was known to normalize the raw data. The second set of scores was made up "artificially," using the McCall technique. The results of comparing these 2 sets of scores are shown in FIGURES 3, 4, 5, and 6. It may be seen that "artificially" normalized scores converge rapidly upon

TABLE 5

DEGREES OF SKEWNESS (G_1) AND KURTOSIS (G_2) FOR HYPOTHETICAL AND ACTUAL DISTRIBUTIONS USED TO COMPARE MCCALL'S T-SCORING METHOD WITH A NORMALIZING LOGARITHMIC TRANSFORMATION*

	Untransformed distribution				*Transformed distribution*			
N	g_1	g_2	g_1/SE_{g_1}	g_2/SE_{g_2}	g_1	g_2	g_1/SE_{g_1}	g_2/SE_{g_2}
10	1.5	1.1	2.2	0.8	1.2	0.6	1.8	0.4
20	1.1	0.4	2.2	0.4	0.7	−0.1	1.4	−0.1
30	1.0	0.4	2.2	0.5	0.6	−0.04	1.4	−0.1
40	0.8	0.7	2.1	0.9	0.2	0.3	0.7	0.4
60	0.8	0.7	2.5	1.1	0.4	0.3	1.2	0.5
120	0.5	0.2	2.5	0.5	0.2	−0.03	0.7	−0.1

*For convenience of presentation, the figures are rounded to 1 decimal place where possible.

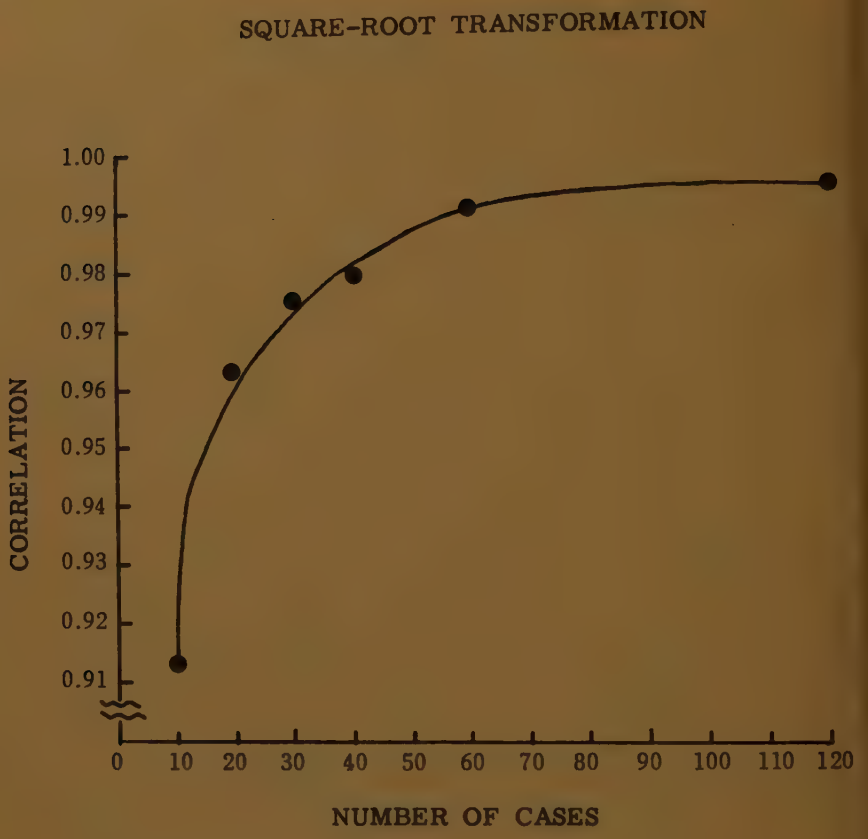

FIGURE 3. Coefficient of correlation, as a function of sample size, between physiological levels expressed in σ units based on a square-root transformation that has been demonstrated (see TABLE 4) to normalize the distribution, and levels expressed in σ units based on McCall's transformation. A curve has been drawn by visual inspection to suggest the form of the relationship. Note that the results of the 2 transformations rapidly converge on each other.

scores normalized by a mathematical transformation. *N*'s of 40 give almost as good results as *N*'s of 120.

These figures, however, do not make one point clear. The greatest differences between the 2 sets of scores invariably occur at the extremes of the distributions. The reason is that McCall's technique truncates the distributions at both ends. The results as a whole suggest that the use of McCall's T-scores can be safely recommended for data such as we obtain, even with relatively small *N*'s, although one has to be careful in certain applications in evaluating extremely high or extremely low reactions.

It should be noted, before leaving this topic, that intraindividual

regression analysis of data obtained in serial experiments requires that several responses be obtained at each of many prestimulus values and that account be taken of the correlation of the components of response with time. In many lengthy serial experiments, it will be found that a process of adaptation sets in that is revealed partially by a monotonically (or almost monotonically) increasing or decreasing prestimulus level. Thus, afterwhile, each succeeding response in the series is elicited from successively higher or lower prestimulus values. This phenomenon of

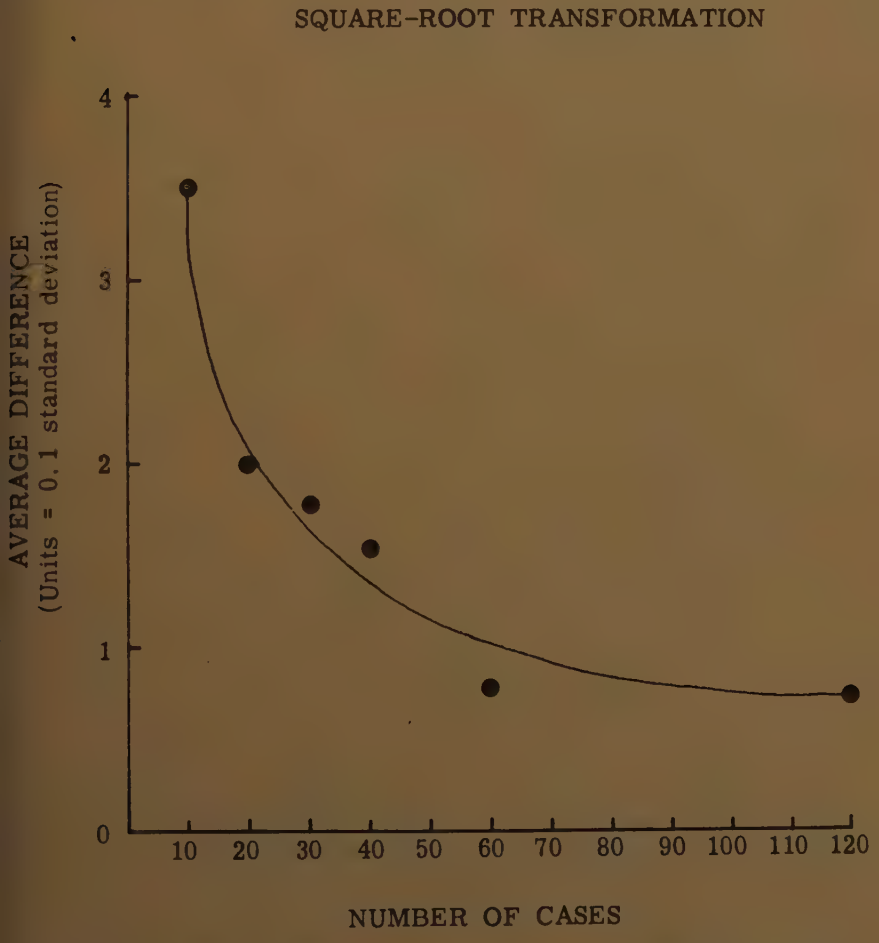

FIGURE 4. Average difference, as a function of sample size, between physiological levels expressed in σ units based on a square-root transformation that has been demonstrated (TABLE 4) to normalize the distribution, and levels expressed in σ units based on McCall's transformation. A curve has been drawn by visual inspection to suggest the form of the relationship.

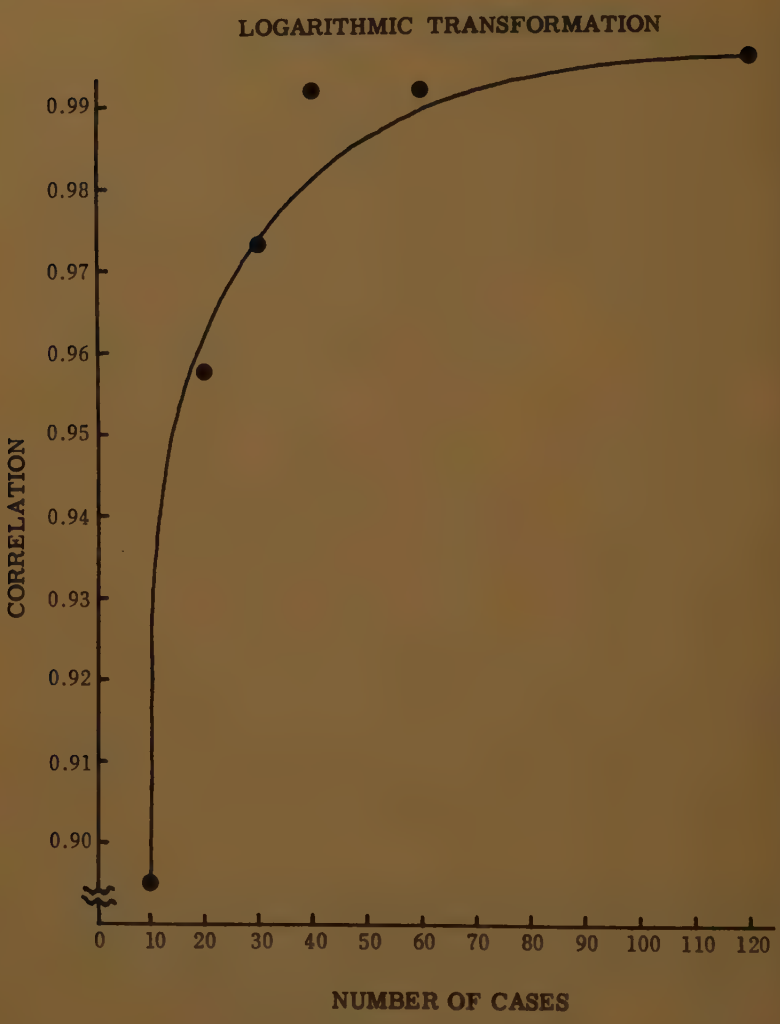

FIGURE 5. Coefficient of correlation, as a function of sample size, between physiological levels expressed in σ units based on a logarithmic transformation that has been demonstrated (TABLE 5) to normalize the distribution, and levels expressed in σ units based on McCall's transformation. A curve has been drawn by visual inspection to suggest the form of the relationship. Note that the results of the 2 transformations rapidly converge on each other.

almost perfect correlation between prestimulus value and time is seen frequently in conditioning experiments when skin-resistance responses are measured. The technique of intraindividual regression analysis is inapplicable in such circumstances, of course. Some other technique of analysis will have to be devised to suit each experiment.

Individual differences in homeostatic efficiency. It may have occurred to the reader that a physiological interpretation of the "law" of initial values, such as we have proposed here, generates a new question and a new vantage point from which individual differences in autonomic reactivity may be surveyed. The question was foreshadowed in FIGURES 1 and 2, where tremendous variation among individuals can be seen in the magnitude of the negative correlation between prestimulus values of heart rate and stimulus-produced changes. Does this variation reflect individual differences in homeostatic efficiency, or is it mere sampling variation?

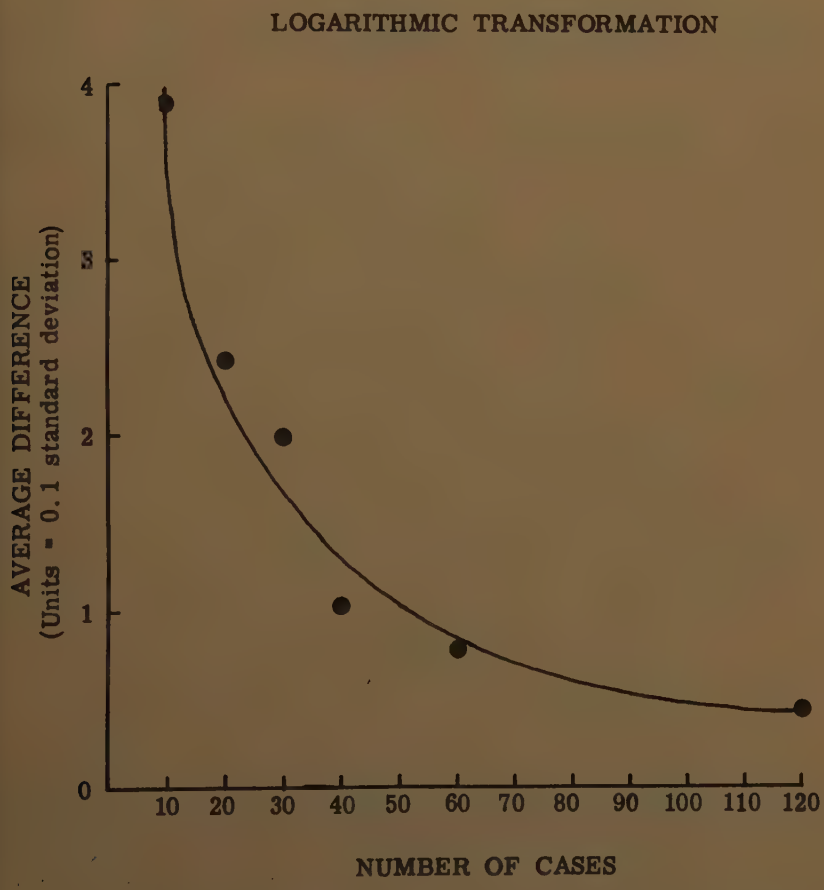

FIGURE 6. Average difference, as a function of sample size, between physiological levels expressed in σ units based on a logarithmic transformation that has been demonstrated (TABLE 5) to normalize the distribution, and levels expressed in σ units based on McCall's transformation. A curve has been drawn by visual inspection to suggest the form of the relationship.

A fresh grasp on this question is provided by some results obtained in as-yet-unpublished studies on semantic conditioning, which are continuations of the studies already cited. In these new experiments we elicit serially 52 heart-rate responses by combined mental-motor activity, in an anxiety-conditioning experiment. FIGURE 7 presents a frequency distribution, for 56 subjects, of individually computed correlations

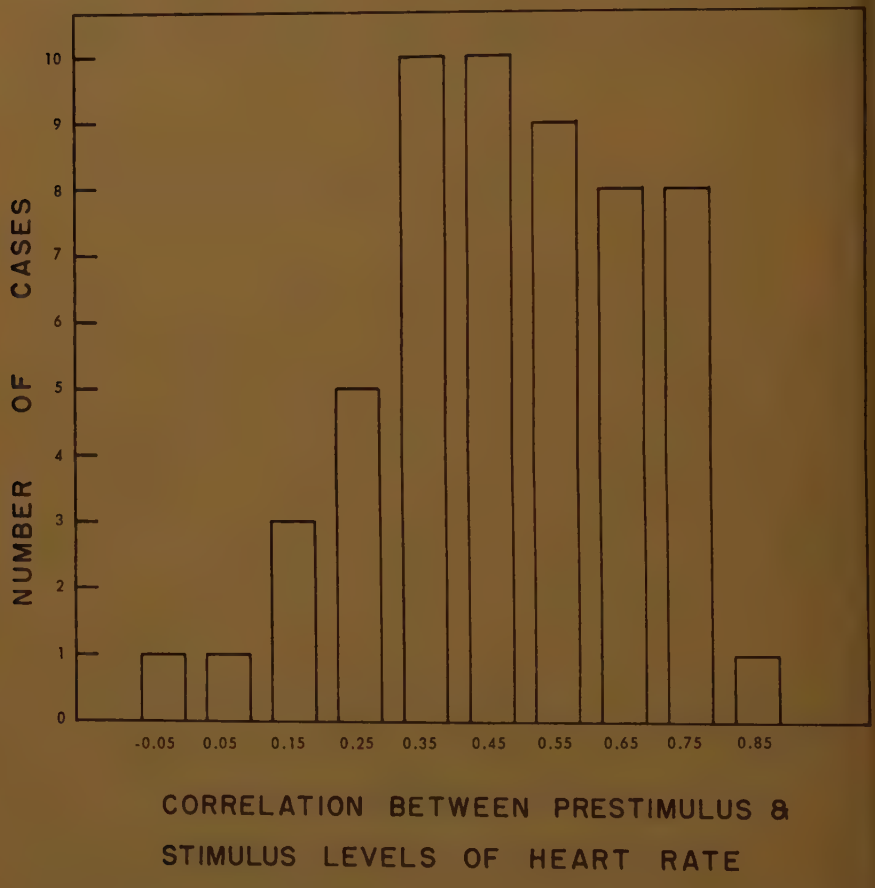

FIGURE 7. Frequency distribution for 56 cases of the individual product-moment coefficients of correlation between prestimulus values of heart rate and stimulated values of heart rate due to combined verbal-motor activity. Distributions of heart rates for each individual have been normalized by McCall's transformation. Each correlation is based on 52 serially elicited reactions. The cardiac activity during each episode of stimulation is measured by averaging the 6 fastest cardiac rates in 15 seconds of combined activity, and is correlated with the average of the 6 fastest beats in the last 10 seconds of the preceding 30-second intertrial rest period. Note the large individual differences in homeostatic efficiency. The rank-order split-half reliability of the correlations is +0.60, uncorrected (see text).

between initial level and stress level using McCall's normalizing technique. Again, tremendous variation is shown, with the correlations falling into class intervals whose midpoints vary from −0.05 to +0.85. Some individuals seem like automatons. The level their heart rate reaches during experimental stimulation is closely related to the prestimulus heart rate. Other individuals seem much more variable; their cardiac rates during stimulation are unrelated to the background level of autonomic excitation; each succeeding episode of verbal-motor activity is responded to almost uniquely.

To study the matter further we selected a sample of 20 individuals, all of whom were in the same experimental group (there are 4 such groups, with systematic variations in experimental treatment among them). The individually computed correlations of these individuals ranged from +0.19 to +0.79. The pool of 52 serial reactions for each individual was split into 2 halves, which were equated for temporal position and semantic connotation. Two correlations were then computed for each subject, 1 for each equivalent subgroup of 26 responses. These pairs of correlations were then themselves correlated by the rank-difference technique. The resulting correlation was +0.60, significant at beyond the 0.01 level. Within this single experimental situation, then, the variation of homeostatic correlations (as we may call them) is not entirely random. There are reliable and systematic individual differences.

In these data, there is a possibility of some circularity of reasoning. The individual correlations were obtained in a study of the growth and decay of conditioned autonomic responses. Perhaps it is precisely those individuals who exhibited the greatest conditioning and generalization who show low correlations. This would follow if the increasing autonomic activation seen as the conditioning proceeded was very marked in absolute terms. If, for example, before conditioning, heart-rate changes were of the order of magnitude of 5 beats/minute, but during and after conditioning were of the order of 30 beats/minute, a low correlation between prestimulus and stimulus values might be produced as a consequence of the very psychological process we are studying. That this potential circularity of reasoning does not exist is suggested by the fact that the quantitative variation of response is not nearly so marked as in the hypothetical example cited above, and by the details of the arithmetical procedure by which we secure indices of conditioning and generalization (see references given). We attacked the problem directly, however, by analyzing curves of conditioning and generalization as a function of the "homeostatic correlation." No statistically significant differences were obtained. Whatever trend there was, moreover, was in a direction that reassured us there is no circularity in our data. It was the individuals

with higher correlations who seemed to condition and generalize more promptly and extensively.

Further analysis showed that these differences in homeostatic efficiency were not related to (1) the variation among individuals in overall level of heart rates, or (2) the variation among individuals in the variability of prestimulus levels of heart rate. Thus, the magnitude of the homeostatic correlation is not to be attributed to variations in the background level of autonomic excitation (such that high correlations would be found in individuals whose heart rates were high, thus more urgently invoking homeostatic restraint).

We tentatively conclude that the apparent and large individual differences in homeostatic efficiency are not attributable to chance variation. They seem real, and it seems likely that the set of individual differences so revealed will turn out to be of some importance. The sources of the variation and the physiological and behavioral consequences need to be ascertained.

All this, taken as a whole, gives a first answer to our original question. We are justified in comparing the reactivity of groups, or of individuals, or of a given individual to varying stimuli, only to the degree to which we realize and account for the fact that the autonomic reaction we record is a complex manifestation of opposing influences. A rational solution to one important problem posed by the existence of such homeostatic mechanisms – the law of initial values – may be approached by using the regression of stress level of functioning upon prestimulus level of functioning.

This is not the whole story, however. A second and most provocative complication is produced by the fact, only recently established experimentally, that autonomic responses are systematically organized into patterns of activation. Limitations of space forbid any but the briefest review of the facts in this area of investigation.

The Patterning of Autonomic Responses

Interindividual response specificity. The correlations among autonomic responses to stress are usually rather low, indicating either a lack of communality or of reliability. In 5 separate experiments, only 3 of which have been published as yet (J. I. Lacey, 1950; J. I. Lacey and R. Van Lehn, 1952; J. I. Lacey, D. E. Bateman, and R. Van Lehn, 1953), we have found strong evidence that the lack of correlation is due to systematic and idiosyncratic patterns of response. The main facts are well illustrated in FIGURE 8, which shows the responses of a 45-year-old woman to 3 different stresses. These are autonomic lability scores

rranged in T-score form with a mean of 50 and a standard deviation of 0. The graph illustrates 2 phenomena:

(1) A marked intraindividual differentiation of autonomic response. Whether we call this person markedly overreactive, markedly underreactive, or of average reactivity, depends on the function being measured.

(2) This individual shows what we have called *relative response specificity*. Whatever the stress, she tends to respond with the same hierarchy of activation.

Statistical and experimental analyses of various sorts show in various ways that this is a reliable phenomenon. Individuals tend to respond to different stresses, which impose varying physiological and psychological demands on the organism, with reproducible patterns of autonomic activation. Over a period of 4 years, moreover, the patterns of response to the cold-pressor test are highly reproducible. What is not

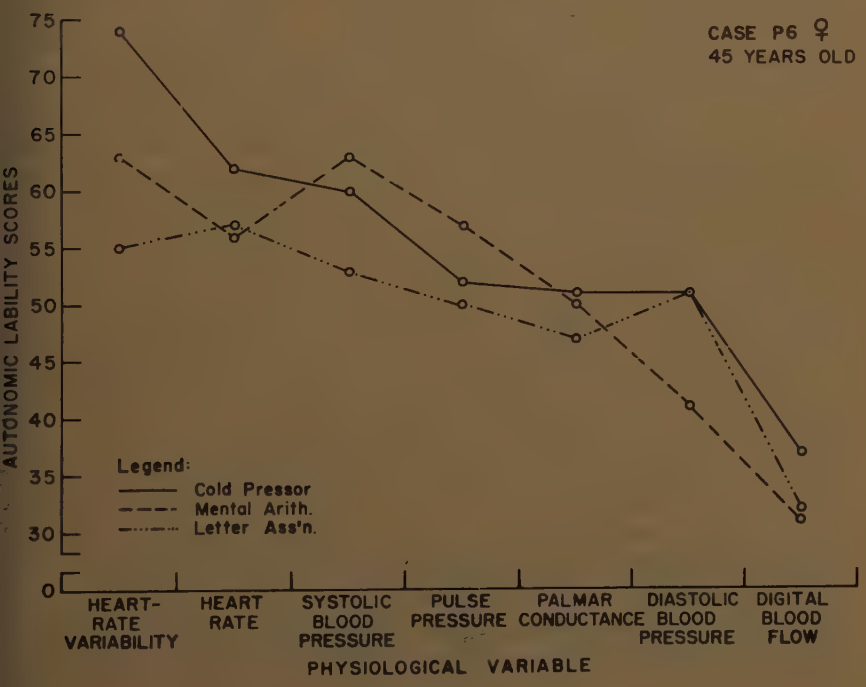

FIGURE 8. Profile of the autonomic activation for a 45-year-old woman who was subjected serially to 3 stresses: cold-pressor test, mental arithmetic, and letter association. Autonomic reactions are expressed by autonomic lability scores (see text). Note the marked intraindividual differentiation of autonomic response. The woman is markedly overreactive in heart rate and heart-rate variability, for example, but markedly underreactive in digital blood flow. Note also that she exhibits "relativeresponse specificity." The general pattern of activation is the same from stress to stress.

yet clear is whether these results are a statistical expression of a tendency common to all people to yield idiosyncratic patterns of response, or whether there are quantitative variations in this tendency towards differentiation and stereotypy of patterns of activation. On general biological grounds, and on the basis of our data to date, we favor the latter hypothesis, and we are undertaking experiments to test it. However this latter hypothesis turns out, it is clear that intraindividual differentiation of autonomic response is a factor to be considered. Both the direction and degree of differentiation among individuals depends on the physiological function measured. Using one physiological measure, we should claim that *A* is less reactive than *B*; using another, we should claim the reverse. Corroboration of this principle is provided by Terry (R. A. Terry, 1953) and, for the case of individuals with frank psychosomatic disorders, by Malmo and his collaborators (R. B. Malmo, C. Shagass, and F. H. Davis, 1950), who independently formulated a closely related principle of "symptom specificity."

One possible way to rescue a concept of over-all autonomic reactivity is indicated in a previous publication (J. I. Lacey, D. E. Bateman, and R. Van Lehn, 1952). We were there able to show a significant relationship between the Rorschach form-color index of "emotionality" and autonomic responses to stress, only if the individual's reactivity was assayed in terms of his own pattern of response. Two individuals, in other words, who exhibited a maximum autonomic lability score of 62, let us say, were called equally reactive, although 1 individual showed his maximal score of 62 in heart rate, and the other in palmar conductance. This specific relationship, however, was not reproducible in an unpublished study by Vaughn Crandall and myself on a different population of individuals. Obviously, much study and thought will have to be given to the problem of evaluating such patterns.

Interstressor response specificity. The pattern of autonomic activation is different in different affective states. Thus, Ax (A. Ax, 1953) has been able to show physiological differentiation between responses he labeled "anger" and "fear." Funkenstein and his collaborators have shown differences in the patterns of arousal in responses labeled "anger-out," "anger-in," and "anxiety" (D. H. Funkenstein, S. H. King, and M. Drolette, 1954). The relationship between these findings and the principles of "symptom specificity" and "relative response specificity" is still unclear. A direct and quantitative attack on the problem of what proportion of the variation in response is to be attributed to individual differences in the organization of the autonomic nervous system and what proportion is to be attributed to the nature of the individual's psychological mechanisms of coping with specific stressor episodes has yet to be made.

Summary and Conclusions

Now let us retrace, in a summary, the long and rather involved path own which we have come. We have shown, for a variety of populations nd stresses and for cardiovascular and sudomotor variables, that the agnitude of stimulus-produced activation of the autonomic nervous ystem, in general, is related negatively to the prestimulus level of physio- ogical function. We have argued that this is not simply an arithmetical rtifact of there being but a limited range of response left available to an rganism already functioning at high levels of autonomic excitation, but hat it is a physiological consequence of, and deducible from, the facts f homeostatic restraint of response. We believe that the same mecha- isms operate for variables other than those of the cardiovascular and udomotor, and we have briefly touched upon the relevant literature.

We have proposed that arbitrary and empirical statistical transforma- ions causing this phenomenon to disappear within a given body of ex- erimental data are irrational procedures, and that the rational procedure s to establish statistical norms, that is, frequency distributions of esponses at varying prestimulus levels of functioning for varying popu- ations and stresses. Since this is manifestly impossible, we propose hat the statistical best approximation to this ideal solution is attained y use of the technique of regression analysis.

We then attempted to demonstrate that removing the regression of lgebraic or percentage response on initial level was spurious, redundant, nd awkward. We proposed then to analyze only the regression of stress evel on initial level. The scores resulting from such a regression analy- is we called autonomic lability scores.

This technique proved adequate in all respects, and it yielded clear- y interpretable results. The technique, moreover, freely allows "para- oxical" phenomena to be exhibited, that is, an apparent lack of auto- omic increment or the appearance of autonomic decrement when experi- ental stimulation is imposed upon high background autonomic excita- ion. Such "paradoxical" events may receive high autonomic lability cores in the proposed regression model.

We then illustrated the utility of autonomic lability scores in adding larity of interpretation to some data on maturational changes in patterns f autonomic activation.

The concept and the measure were then extended to the individual ase. One difficulty in this application appeared in that nonnormal dis- ibutions (within individuals) were found. The applicability of an "arti- cial" normalizing technique (McCall's T-scores) was investigated by a mited empirical study. The results suggested that such an "artificial" echnique may be used safely with the sort of data we obtain.

It was shown that both the viewpoint and the data generated new questions and new phenomena: Are there reliable individual differences in homeostatic efficiency revealed by the coefficient of correlation between prestimulus and stimulus levels of autonomic excitation? The data presented tentatively suggest an affirmative answer.

Finally, we briefly considered the fact that autonomic activation does not occur as a quantitatively undifferentiated and mass discharge. Instead, it is patterned. There are both reliable interindividual and reliable interstressor differences in the patterns of activation.

It is clear, then, that there are 3 aspects to the answer to our original question. To evaluate autonomic responsivity we must (1) take the law of initial values into account, (2) choose autonomic variables that are relevant to the individual, and (3) choose variables that are relevant to the behavioral sequence we are studying. For the first problem, the elements of a general concept applicable to the formulation of a general solution are at hand. In facing the problems of patterned activation of the autonomic nervous system, we are at the very edge of knowledge. A vast area of ignorance faces us, to challenge the efforts of psychologist, physiologist, and physician, of clinician and laboratory scientist.

APPENDIX

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To find the correlation between the variable x and the variable $\lambda = (y\text{-}x)$, with x and y expressed as raw scores, let $\bar{x}$, σ_x, $\bar{y}$, σ_y and r_{xy} be the parameters of the bivariate normal distribution of x and y, and let E stand for the "expected" or mean value. Then, from the standard expression for the correlation coefficient:

$$(1)\quad r_{x\lambda} = \frac{E[x(y\text{-}x)] - E(x)\,E(y\text{-}x)}{\sigma_x\,\sigma_\lambda}$$

Now $E[x(y\text{-}x)] = E(xy - x^2) = E(xy) - E(x^2)$,

$E(x) = \bar{x}$, and

$E(y\text{-}x) = E(y) - E(x) = (\bar{y}\text{-}\bar{x})$.

Then, $E[x(y\text{-}x)] - E(x)\,E(y\text{-}x) = E(xy) - E(x^2) - \bar{x}\,\bar{y} + \bar{x}^2$

$= [E(xy) - \bar{x}\,\bar{y}\,] - [E(x^2) - \bar{x}^2]$

$= r_{xy}\,\sigma_x\,\sigma_y - \sigma_x^2$.

And, as is well known,

$$\sigma_{(y\text{-}x)} = \sqrt{\sigma_y^2 - 2r_{xy}\,\sigma_x\,\sigma_y + \sigma_x^2}$$

ence:

$$(2)\quad r_{x\lambda} = \frac{r_{xy}\,\sigma_x\,\sigma_y - \sigma_x^2}{\sigma_x\sqrt{\sigma_y^2 - 2r_{xy}\,\sigma_x\,\sigma_y + \sigma_x^2}}$$

$$= \frac{r_{xy}\,\sigma_y - \sigma_x}{\sqrt{\sigma_y^2 - 2r_{xy}\,\sigma_x\,\sigma_y + \sigma_x^2}}$$

r, letting $\phi = \sigma_x/\sigma_y$, and substituting $\sigma_y\phi$ for σ_x in FORMULA 2, we get

$$(3)\quad r_{x\lambda} = \frac{r_{xy}\,\sigma_y - \sigma_y\phi}{\sqrt{\sigma_y^2 - 2r_{xy}\,\sigma_y^2\phi + \sigma_y^2\phi^2}}$$

$$= \frac{r_{xy} - \phi}{\sqrt{1 - 2r_{xy}\,\phi + \phi^2}}$$

ince the denominator is $\sigma_{(y-x)}/\sigma_y$, and consequently positive, the cor-ect value of the square root is the positive one. Hence, if $r_{xy} < \phi$, that is, f $r_{xy} < \sigma_x/\sigma_y$, $r_{x\lambda}$ will be negative; if $r_{xy} > \phi$, $r_{x\lambda}$ will be positive. If $_{xy} = 0$, $r_{x\lambda}$ will clearly not be zero, but will equal $-\phi / \sqrt{1+\phi^2}$ or $\sigma_x/\sqrt{\sigma_y^2+\sigma_x^2}$. A related formula for the correlation between x and $y + x$ s given by Snedecor (G. Snedecor, 1946, p. 163). Returning now to ORMULA 2, we have, by a series of simple steps:

$$r_{x\lambda} = \pm\frac{\sqrt{r_{xy}^2\,\sigma_y^2 - 2r_{xy}\,\sigma_y\,\sigma_x + \sigma_x^2}}{\sqrt{\sigma_y^2 - 2r_{xy}\,\sigma_x\sigma_y + \sigma_x^2}}$$

$$= \pm\frac{\sqrt{(\sigma_y^2 - 2r_{xy}\,\sigma_y\sigma_x + \sigma_x^2) + (r_{xy}^2\,\sigma_y^2 - \sigma_y^2)}}{\sigma_y^2 - 2r_{xy}\,\sigma_x\sigma_y + \sigma_x^2}$$

$$= \pm\sqrt{1 - \frac{\sigma_y^2\,(1 - r_{xy}^2)}{\sigma_y^2 - 2r_{xy}\,\sigma_x\sigma_y + \sigma_x^2}}$$

which

; the result given in the preceding article. The sign of the square root ; given by the sign of the quantity $(r_{xy} - \phi)$.

To discuss the correlation between x and the variable $\theta = (y\text{-}x)/x$, we proceed as follows. Let x and y be as above, let $V = \sigma_x/\bar{x}$ be the coefficient of variation of x, and $W = \sigma_y/\bar{y}$ be the coefficient of variation of y. We assume V and W to be sufficiently small to permit ignoring powers higher than the fourth of V, W, and their products, in what follows. It is probable that with V and W no greater than 25 per cent, the resulting error in $r_{x\theta}$ will be no more than 2 per cent. Then, as we shall show, the correlation $r_{x\theta}$ is given by:

$$(4) \quad r_{x\theta} = \frac{A\sqrt{1+3V^2}}{\sqrt{\frac{1+8V^2}{1+3V^2} + \frac{1-r_{xy}^2}{\left(\frac{V}{W}-r_{xy}\right)^2}}}$$

where $A = -1$ if $(V - r_{xy}W)$ is positive, $+1$ if $(V - r_{xy}W)$ is negative. [A more approximate formula is given by Snedecor (G. Snedecor, 1946, p. 164). The sign of the middle term of the denominator in Snedecor's formula, however, appears to be in error.]

Clearly, it is the quantity $\left[\frac{V}{W} - r_{xy}\right]$ rather than r_{xy} itself which plays the dominant role in determining the value of $r_{x\theta}$. This fact can be pointed up by considering a few special cases.

For example, if $r_{xy} = 1$, $r_{x\theta}$ will be approximately equal to -1 if $V > W$, or to $+1$ if $W > V$, or (exactly) to 0 if $W = V$. If $r_{xy} = 0$, $r_{x\theta}$ is almost certainly not zero, rather being equal to

$$r_{x\theta} = \frac{-\sqrt{1+3V^2}}{\sqrt{\frac{1+8V^2}{1+3V^2} + \frac{(W)^2}{(V)^2}}} \sim \frac{-1}{\sqrt{1+\frac{W^2}{V^2}}}$$

When $V = W$, $r_{x\theta}$ becomes

$$r_{x\theta} = \frac{-\sqrt{1+3V^2}}{\sqrt{\frac{1+8V^2}{1+3V^2} + \frac{1+r_{xy}}{1-r_{xy}}}} \sim -\sqrt{\frac{1-r_{xy}}{2}}$$

To simplify the derivation of FORMULA 4, let E represent, as above, the expected or mean value, let $x = \bar{x}(1+u)$, $y = \bar{y}(1+v)$. Then $E(u) = E(v) = 0$, $E(u^2) = V^2$, $E(v^2) = W^2$. Further, $r_{uv} = r_{xy}$, because u is a linear transform of x, and v of y.

Since $(1 + u)$ is also a linear transform of x and since, because:

$$\theta = (y\text{-}x)/x = y/x - 1 = \frac{(\bar{y})}{(\bar{x})}\frac{(1+v)}{(1+u)} - 1, \ \theta \text{ and } (1+v)/(1+u)$$

e transforms of each other, the correlation between x and θ is identical th that between $(1 + u)$ and $(1 + v)/(1 + u)$.

ence:

$$(5) \qquad r_{x\theta} = \frac{E\left[(1+u)\cdot\frac{(1+v)}{(1+u)}\right] - E\,(1+u)\,E\,\frac{(1+v)}{(1+u)}}{\sigma(1+u)\ \sigma\frac{(1+v)}{(1+u)}}$$

l but 2 of these terms can be evaluated quickly:

$$(6) \qquad E\left[(1+u)\frac{(1+v)}{(1+u)}\right] = E\,(1+v) = 1$$

$$(7) \qquad E\,(1+u) = 1$$

$$(8) \qquad \sigma(1+u) = \sigma_u = V$$

For the other terms, we make use of the following expansions, ob- ined by use of the binominal expansion theorem:

$$(9) \qquad 1/(1+u) = (1+u)^{-1} = 1 - u + u^2 - u^3 + u^4 - \ldots$$

$$(10) \qquad 1/(1+u)^2 = (1+u)^{-2} = 1 - 2u + 3u^2 - 4u^3 + 5u^4 - \ldots$$

Thus, using FORMULA 9 we get:

$$(11) \qquad E\frac{(1+v)}{(1+u)} = E\left[(1+v)\frac{(1)}{(1+u)}\right] = E\left[(1+v)(1-u+u^2-u^3+u^4-\ldots)\right]$$

$$= E\left[(1-u+u^2-u^3+u^4-\ldots)+(v-uv+u^2v-u^3v+u^4v-\ldots)\right]$$

$$= E\,(1) - E\,(u) + E\,(u^2) - E\,(u^3) + E\,(u^4) + E\,(v) - E\,(uv) + E\,(u^2v) - E\,(u^3v) + E\,(u^4v) - \ldots$$

$$= 1 - 0 + V^2 - 0 + 3V^4 + 0 - r_{xy}\,VW + 0 - 3r_{xy}\,V^3W + 0 - \ldots$$

$$= 1 + V^2 + 3V^4 - r_{xy}\,VW - 3r_{xy}\,V^3W - \ldots$$

The evaluation of the individual expected values above (and of those ow) follows the formulas given by Kendall (M. G. Kendall, 1943, p. 89, tion 3.15; and p. 129, formula 5.52).

Using FORMULA 10, we get

$$(12)\quad E\left[\frac{1+v}{1+u}\right]^2 = E\,(1+v)^2\left(\frac{1}{(1+u)}\right)^2 = E\left[(1+2v+v^2)\,(1-2u+3u^2-4u^3+5u^4-\ldots)\right.$$

$$= E\;(1-2u+3u^2-4u^3+5u^4-\ldots)+2(v-2uv+3u^2v-4u^3v+5u^4v-\ldots)$$
$$+\,(v^2-2uv^2+3u^2v^2-4u^3v^2+5u^4v^2-\ldots)$$

$$= E\,(1)-2E(u)+3E(u^2)-4E(u^3)+5E(u^4)+2E(v)-4E(uv)+$$
$$6E(u^2v)-8E(u^3v)+10E(u^4v)+E(v^2)-2E(uv^2)+3E(u^2v^2)$$
$$-4E(u^3v^2)+5E(u^4v^2)-\ldots$$

$$= 1-0+3V^2-0+5\cdot 3V^4+0-4r_{xy}VW+0-8\cdot 3r_{xy}V^3W+0+$$
$$W^2-0+3(1+2r_{xy}^2)\,V^2W^2-0+5\cdot 3\,(1+4r_{xy}^2)\,V^4W^2$$

$$= 1+3V^2+15V^4-4r_{xy}VW-24r_{xy}V^3W+W^2+3(1+2r_{xy}^2)\,V^2W^2$$
$$+\,15\,(1+4r_{xy}^2)\,V^4W^2+\ldots$$

Combining FORMULAS 11 and 12, we find (ignoring all terms for which the combined exponents of V and W exceed 4) that:

$$(13)\quad \sigma^2\frac{(1+v)}{(1+u)} = E\left[\frac{(1+v)}{(1+u)}\right]-\left[E\frac{(1+v)}{(1+u)}\right]^2$$

$$=1+3V^2+15V^4-4r_{xy}VW-24r_{xy}V^3W+W^2+3(1+2r_{xy}^2)\,V^2W^2+\ldots$$
$$\left[1+2V^2+7V^4-2r_{xy}VW+r_{xy}^2\,V^2W^2-8r_{xy}V^3W\right]$$

$$= V^2+W^2-2r_{xy}VW+8V^4-16r_{xy}V^3W+3V^2W^2+5r_{xy}^2\,V^2W^2$$

$$= (1+8V^2)\,(V^2-2r_{xy}VW)+(1+3V^2)W^2+5r_{xy}^2\,V^2W^2$$

$$= (1+8V^2)\,(V^2-2r_{xy}VW+r_{xy}^2W^2)+(1+3V^2)\,(1-r_{xy}^2)W^2$$

$$= (1+8V^2)\,(V-r_{xy}W)^2+(1+3V^2)\,(1-r_{xy}^2)W^2$$

Substituting the values of FORMULAS 6, 7, 8, 11, and 13 in FORMULA 5, we get (ignoring higher powers of V and W):

$$(14)\quad r_{x\theta} = \frac{1-\left[1+(1+3V^2)\;(V^2-r_{xy}VW)\right]}{V\sqrt{(1+8V^2)\,(V-r_{xy}W)^2+(1+3V^2)(1-r_{xy}^2)W^2}}$$

$$= \frac{-(1+3V^2)\,(V-r_{xy}W)}{\sqrt{(1+8V^2)\,(V-r_{xy}W)^2+(1+3V^2)(1-r_{xy}^2)W^2}}$$

viding numerator and denominator by $(\sqrt{1+3V^2})\ (V-r_{xy}W)$ gives

$$(14)'\quad r_{x\theta} = \frac{A\sqrt{1+3V^2}}{\sqrt{\dfrac{1+8V^2}{1+3V^2} + \dfrac{(1-r_{xy}^2)W^2}{(V-r_{xy}W)^2}}}$$

here $A = -1$ if $V > r_{xy}W$, $A = +1$ if $V < r_{xy}W$. The function of A is to ve FORMULA 14′ the same sign as FORMULA 14. The positive values e always assumed for the square roots.

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